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Emergency department crowding is associated with 28-day mortality in community-acquired pneumonia patients

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Summary *Object:* Although emergency department (ED) crowding has been shown to be associated with delayed antibiotics treatment in community-acquired pneumonia (CAP) patients, association between ED crowding with mortality has not been investigated. We hypothesized emergency department crowding is associated with 28-day mortality in CAP patients.
Methods: A retrospective observational study using prospective database was performed on CAP patients who visited a single, urban, tertiary care hospital ED between April 1, 2008 and September 30, 2009. Main outcomes were 28-day mortality and timeliness of antibiotic therapy (within 2, 4, 6, and 8 h of arrival). ED crowding was measured by Emergency Department Occupancy (EDO) rate. A multivariate logistic regression was performed to determine the association of 28-day mortality with EDO rate after adjusting for factors such as time-to-first-antibiotic-dose (TFAD), pneumonia severity index and laboratory markers.
Results: 477 cases were enrolled during the study period. 28-day mortality rate was 4.8%. EDO rate ranged from 37.2% to 162.8%, and median was 97.7% (IQR: 80.2%–116.3%). When categorized into tertiles by EDO rate, high crowding condition (EDO rate >109.3%) was significantly associated with a higher 28-day mortality (adjusted OR = 9.48, 95% CI 1.53–58.90). However, TFAD was not associated with 28-day mortality. ED crowding was not associated with delay of TFAD at various time intervals (2, 4, 6, and 8 h).

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Conclusions: ED crowding measured by EDO rate was associated with higher 28-day mortality in CAP patients after adjusting TFAD, pneumonia severity index (PSI), and laboratory markers, although there was no association between ED crowding and TFAD.

Introduction

Background

Because emergency department (ED) crowding has been reported to cause delays in diagnosis and treatment, decrease of quality-of-care, and poor outcomes,[1,2] ED crowding has been highlighted as a global health problem. Many crowding scales have been developed such as the National Emergency Department Overcrowding Scale (NEDOCS),[3] the Emergency Department Work Index (EDWIN),[4] the Real-time Emergency Analysis of Demand Indicator (READI),[5] the Emergency Department Crowding Scale (EDCS)[6] and the Work Score (WS).[7] However, there is no agreement as to which definition of ED crowding should be used. Moreover, these scales have limitations to quantifying real-time overcrowding because many EDs are not equipped with electronic tracking systems to calculate these scales in real-time.[8,9] Recently, one simple measure, Emergency Department Occupancy (EDO) rate was conceptualized.[10] Using the concept of 'occupancy' alone, not exact EDO, two Australian studies showed an association between increased mortality and ED crowding.[11,12] Considering its simplicity, calculating the EDO rate and the real-time quantification of ED crowding by this scale seems to be easy for many EDs.

Importance

For community-acquired pneumonia (CAP) patients, recent studies have shown that ED crowding is associated with a delayed time to first antibiotic dose (TFAD).[13–15] However, it is uncertain whether ED crowding is associated with poor outcomes in CAP patients. Thus, study revealing the impact of ED crowding on the mortality in CAP patients is needed.

Goal of this investigation

The purpose of this study was to determine whether there is an association between ED crowding as measured by the EDO rate and mortality in patients who present at the ED with CAP. We hypothesized that ED crowding using the EDO rate is associated with increased mortality in CAP patients.

Methods

Study design and setting

This study was approved by the Institutional Review Board (IRB) of the study hospital, and a waiver for consent was given. We performed a retrospective observational study. This study was part of a prospective quality improvement study to implement Pneumonia Severity Index (PSI) in the admission protocol.[16] IRB number is B-1103/123–104.

The study was conducted at a 950-bed urban academic tertiary care hospital with an annual ED census of 67,000. The ED is staffed by board-certified emergency medicine physicians and rotating residents supervised by these physicians. There was no change in the number of ED staff during the study period. The study hospital is a paperless institution where all medical records are fully electronic.

The ED of the study hospital has 50 beds for patients: 40 beds for adult patients and 10 beds for children. Additionally, 3 beds in the hallway can be used for adult patients. Thus, we used 43 as the total number of beds for adult patients in this ED. The study hospital divides adult and pediatric patients at age 15 and uses the Emergency Severity Index (ESI) as a triage tool in the ED to offer different medical services according to severity of the condition.

Selection of participants

The diagnostic criteria for CAP included the following: 1) respiratory symptoms such as cough, sputum, dyspnea and pleuritic chest pain, 2) abnormal lung sounds such as crackles on physical examination and 3) chest X-ray abnormalities such as infiltration, haziness, consolidation and associated pleural effusion. Fever at presentation was not essential to diagnose CAP. First-line antibiotics for CAP patients were selected according to the 2007 version of Infectious Diseases Society of America/American Thoracic Society (IDSA/ATS) consensus guidelines.[17]

All adult CAP patients diagnosed at the ED between April 1, 2008 and September 30, 2009 were eligible. We excluded the following patients; 1) patients less than 18 years of age, 2) patients transferred from another facility, including the ED of another hospital, an acute care facility at which the patient was an inpatient or an outpatient, any distinct unit of the hospital other than ED and an ambulatory surgery center, 3) patients who left against medical advice or discontinued care on the day of or the day after arrival.

Outcome measures

The primary outcome measure was 28-day mortality. The EDO rate is defined as the number of patients in the ED divided by number of equipped ED beds.[10] We could track the total number of patients in the ED using our electronic medical record (EMR) system (BEST Care, ezCaretech, Co, Ltd, Seoul, Korea). Each CAP patient was matched to the total number of patients in the ED at the time of that patient's arrival at the ED. Then, the EDO rate was calculated by dividing the total number of patients in the ED by the number of equipped ED beds, in this case - 43. Then, we categorized the EDO rate into tertile groups using two cut-off points. The three groups were as follows: the low

crowding group, the intermediate crowding group and the high crowding group.

Data collection and processing

A prospective database was obtained from the previous study.[16] This database includes demographics, clinical data and physical findings. Clinical data included age, sex, and co-morbidities such as hypertension, diabetes mellitus, liver cirrhosis, malignancy state, and congestive heart failure. Physical findings included initial systolic and diastolic blood pressure, heart rate, respiratory rate and body temperature. Laboratory data included complete blood count, chemistry, C-reactive protein (CRP) levels, arterial blood gas analysis, and PaO_2/FiO_2 ratio. PSI was calculated for each patient. Other data including 28-day mortality, the time of ED arrival and the time of first antibiotic dose delivery were also obtained from the database. For CAP patients whose mortalities we did not know from these record because of transfer to other facilities or discharge, our trained abstractor made a phone call to the patient or his/her family to determine the patient's status. After explaining the purpose and process of this study and gaining the approval for participation, we proceeded data collection relating to mortality.

Primary data analysis

First, we used univariate logistic regression analysis to determine the association of 28-day mortality with EDO rate, PSI, TFAD and laboratory factors that are not included in PSI. Because PSI is based on demographic data, and clinical and laboratory findings, the demographic and clinical findings are not analyzed again. A multivariate logistic regression analysis was used to determine the adjusted effects of ED crowding on 28-day mortality, after controlling for factors that showed p-value <0.05 and that were considered to show a trend (p-value <0.10) in the univariate logistic regression analysis. TFAD has been shown to be associated with mortality in CAP patients by a former study; thus, TFAD was included in the multivariate logistic regression analysis.[18]

Second, we investigated the association between TFAD and ED crowding. Previous studies have found that ED crowding affects TFAD.[13–15] TFAD was dichotomized using four different time intervals according to the proposal of the Joint Commission and the Centers for Medicare and Medicaid Services as follows: 8_hour (PN-5a), 6_hour (PN-5c), 4_hour (PN-5b), and 2_hour. For each time interval, univariate and multivariate logistic regression analyses were performed to determine whether there was an association between TFAD and EDO. Cox proportional hazard regression analysis was conducted with identified variables, and Kaplan–Meier survival analysis was performed to show the cumulative survival of each crowding group. A Pearson correlation test was performed to determine the extent of the association between two continuous variables — TFAD and EDO rate.

Continuous data are presented as the median and interquartile range (IQR). Binomial data are presented as the percentage frequency of occurrence. Analyses were conducted using SPSS for Windows 17.0 (SPSS Inc., Chicago, IL). The regression results are expressed as odds ratios (OR) with 95% confidence intervals (CI). p-Values were 2-tailed.

Results

Baseline characteristics

During the study period, 597 patients met the initial eligibility criteria. Among these patients, we then excluded

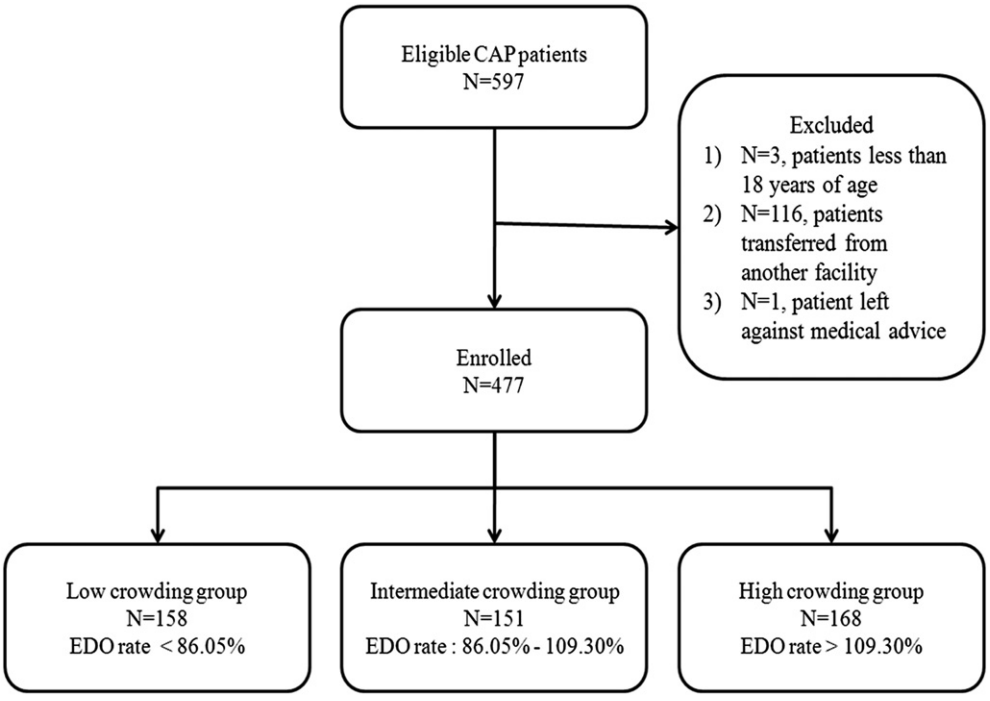

Figure 1 Flow of study patients.

Table 1 Baseline characteristics (median value (IQR) or number (%)).

	Total enrolled group	Low crowding group	Intermediate crowding group	High crowding group	*p*-Value
Number of patients	477	158	151	168	
Age (years)	67 (51.0—76.0)	65 (45.8—76.0)	68 (51.0—77.0)	67 (53.3—75.8)	0.653
Number of Males	268 (56.2)	89 (56.3)	82 (54.3)	97 (57.7)	0.827
SBP[a] (mmHg)	131 (118—147)	121.8 (135.5—150.3)	129 (113—148)	129 (118.3—144)	0.024
HR[b] (/min)	101 (87—114)	102.5 (90—115)	98 (80—111)	101.5 (86—115)	0.051
RR[c] (/min.)	20 (20—22)	20 (20—22)	20 (20—22)	20 (20—22)	0.519
BT[d] (°C)	37.2 (36.6—38.2)	37 (36.5—38.3)	37.1 (36.6—38.1)	36.7 (37.2—38.2)	0.871
Hemoglobin (g/dl)	12.9 (11.7—14.4)	13.0 (11.7—14.5)	12.9 (11.7—14.3)	13.0 (11.7—14.1)	0.438
White Blood Cell (k/μL)	10.6 (7.9—14.5)	10.5 (7.3—14.0)	10.9 (8.4—14.9)	10.9 (8.3—14.4)	0.485
Platelet (k/μL)	257.5 (199.0—330.3)	240 (186.0—293.5)	264 (204.0—346.0)	262.5 (207.3—338.8)	0.183
CRP[e] (mg/dl)	6.6 (2.7—14.5)	6.5 (2.6—14.1)	6.6 (3.0—15.0)	6.6 (2.3—14.0)	0.722
Serum Creatinine (mg/dl)	1.1 (0.8—1.2)	1.0 (0.8—1.2)	1.0 (0.8—1.2)	1.0 (0.9—1.2)	0.683
Albumin (mg/dl)	3.9 (3.5—4.2)	4.0 (3.5—4.3)	3.9 (3.6—4.1)	3.9 (3.5—4.3)	0.532
Total Cholesterol (mg/dl)	162.0 (137.0—188.0)	164 (140.0—191.0)	167 (138.0—192.5)	155.5 (133.0—183.3)	0.022
PaO_2/FiO_2	289.3 (190.7—378.6)	279.3 (181.5—396.7)	291.4 (198.3—377.4)	291.9 (195.8—375.0)	0.810
PSI[f]	74 (51—103)	72.5 (46—104)	75 (52—100)	74 (52—104)	0.628
Number of PSI I	87 (18.2)	34 (21.5)	24 (15.9)	29 (17.3)	0.407
Number of PSI II	132 (27.7)	44 (27.8)	44 (29.1)	44 (26.2)	0.841
Number of PSI III	98 (20.6)	31 (19.6)	34 (22.5)	33 (19.6)	0.770
Number of PSI IV	115 (24.1)	34 (21.5)	36 (23.8)	45 (22.8)	0.539
Number of PSI V	45 (9.4)	15 (9.5)	13 (8.6)	17 (10.1)	0.899
TFAD[g] (min)	166.5 (111.0—279.3)	161.5 (106.5—262.8)	185 (117—313)	164.5 (113—287.8)	0.552
Number of TFAD $<$ 8 h (PN-5a)	380 (79.7)	122 (77.2)	119 (78.8)	139 (82.7)	0.444
Number of TFAD $<$ 6 h (PN-5c)	355 (74.4)	117 (74.1)	110 (72.9)	128 (76.2)	0.786
Number of TFAD $<$ 4 h (PN-5b)	275 (57.7)	94 (59.5)	81 (53.6)	100 (59.5)	0.485
Number of TFAD $<$ 2 h	115 (24.1)	42 (26.6)	35 (23.2)	38 (22.6)	0.671
Mean EDO[h] rate (%)	97.7 (80.2—116.3)	74.4 (65.1—79.7)	95.4 (90.7—102.3)	120.9 (114.0—130.2)	

[a] SBP, systolic blood pressure.
[b] HR, heart rate.
[c] RR, respiration rate.
[d] BT, body temperature.
[e] CRP, C-Reactive Protein.
[f] PSI, Pneumonia Severity Index.
[g] TFAD, Time to First Antibiotic Dose.
[h] EDO, Emergency Department Occupancy.

patients as follows: 1) 3 patients less than 18 years of age, 2) 116 patients transferred from another facility and 3) 1 patient who left against medical advice or discontinued care on the day of or the day after arrival. Finally, 477 patients were enrolled in our study.

The median age was 67 years (IQR: 51—76), and 266 patients (56.2%) were male. Twenty-three patients (4.8%) were 28-day non-survivors after their arrival to the ED. Among the 477 CAP patients enrolled, the mortality status of 6 patients could not be determined (1.3%).

Table 2 Mortality rate by tertile [a]EDO rate group and [b]PSI.

	PSI I	PSI II	PSI III	PSI IV	PSI V
Low crowding group	0/34	0/44	0/31	1/34 (2.9%)	2/15 (13.3%)
Intermediate crowding group	0/24	0/44	0/34	5/36 (13.9%)	4/13 (30.8%)
High crowding group	0/29	0/44	1/33 (3.0%)	4/45 (8.9%)	6/17 (35.3%)

[a] ∫EDO, Emergency Department Occupancy.
[b] PSI, Pneumonia Severity Index.

The median EDO rate was 97.7% (IQR: 80.2%–116.3%). The EDO rate ranged from 37.2% to 162.8%. When categorizing the EDO rates into tertiles, the first cut-off point was 86.1%, and the second was 109.3%. The low crowding group, the intermediate crowding group and the high crowding group were matched to EDO rate levels of <86.1%, 86.1%–109.3%, and >109.3%, respectively (Fig. 1). One hundred fifty-eight patients were assigned to the low crowding group, 151 patients to the intermediate crowding group and 168 patients to high crowding group. Three patients in the low crowding group (1.9%), 9 patients in the intermediate crowding group (6.0%) and 11 patients in the high crowding group (6.6%) died. The baseline characteristics are shown in Table 1.

There were 87 patients in PSI I (18.2%), 132 patients in PSI II (27.7%), 98 patients in PSI III (20.6%), 115 patients in PSI IV (24.1%) and 45 patients in PSI V (9.4%). One patients in PSI III (1.0%), 10 patients in PSI IV (8.7%) and 12 patients in PSI V (26.7%) died respectively. Table 2 shows the mortalities of each crowding group according to the PSI class.

The median TFAD was 166.5 min (IQR: 111.0–279.3). Accomplishments associated with each time interval are shown in Table 1. Although PN-5c (TFAD < 6_hr) and PN-5b (TFAD < 4_hr) were currently used in US, we presented number of TFAD < 8_hr (PN-5a) and number of TFAD < 2_hr together to determine an association with ED crowding and each TFAD time interval.

Logistic regression analysis

In the univariate logistic regression analysis, CRP ($p \leq 0.001$), serum creatinine ($p = 0.008$), albumin ($p \leq 0.001$), PaO_2/FiO_2 ($p \leq 0.001$), and PSI ($p \leq 0.001$) showed significant associations with 28-day mortality. Total cholesterol ($p = 0.052$) and being in the high crowding group ($p = 0.052$) were considered to be trend factors. TFAD ($p = 0.330$) was not a significant factor (Table 3).

To determine the extent of the association between 28-day mortality and ED crowding, a multivariate logistic regression analysis was performed after controlling for significant and trend factors, including the previously conceptualized factor by former studies, TFAD. The high crowding group ($p = 0.016$, adjusted OR = 9.482, 95% CI: 1.526–58.901) along with albumin ($p = 0.002$) and PSI ($p = 0.004$), remained as significant factors (Table 3).

Another multivariate logistic regression analysis was performed to determine whether ED crowding is associated with each time interval of TFAD in same manner as for 28-day mortality. However, we failed to find significant differences in each TFAD time intervals for these cohorts (8_hr < TFAD, 6_hr < TFAD, 4_hr < TFAD and 2_hr < TFAD) (Table 4). The hazard ratio for the high crowding group was 6.391 ($p = 0.018$, 95% CI: 1.369–29.842) (Table 5). The Kaplan–Meier curve for the 28-day cumulative mortality of each crowding group is shown in Fig. 2. The Pearson correlation analysis between TFAD and EDO rate did not yield a significant result (Pearson correlation coefficient = 0.009, p-value = 0.856).

Discussion

We demonstrated that high crowding group was associated with significantly higher mortality than the low crowding group. Surprisingly, there was no significant association between ED crowding and the time to first antibiotic dose delivery.

After it was reported that timely antibiotics administration was associated with the improvement of CAP patient outcomes,[18–20] TFAD has been regarded as a quality-of-care measure. The Hospital Quality Alliance (HQA), the Joint Commission (TJC), the Centers for Medicare & Medicaid Services (CMS), and the National Quality Forum (NQF) published the guideline of delivering the first antibiotic dose within 4 h of hospital arrival as PN-5b.[21–23] In April 2007, the NQF revised its requirement of antibiotic timing from 4 h (PN-5b) to 6 h (PN-5c) to address the unintended consequences of the 4 h time limit. Subsequently, the TJC and CMS also changed their requirement of the time to first antibiotic dose to 6 h in 2007, and PN-5c is currently one of the important measures of a hospital's quality-of-care.

After the change in the required time to first antibiotic dose, studies about CAP and ED crowding have focused on the delay of initial antibiotic delivery, not on mortality.[18,19] In a recent study, EDO rates greater than the median were associated with a decreased OR of receiving antibiotics within 4 h (OR: 0.31, 95% CI: 0.13–0.75).[15] However, previous studies did not report whether mortality in CAP patients was influenced by ED crowding in their cohort. To our

Table 3 Associated factors with 28-day mortality by logistic regression analysis.

Variable	Unadjusted odds ratio	95% CI	p-value	Adjusted odds ratio	95% CI	p-value
Low crowding group	Reference			Reference		
Intermediate crowding group	3.279	0.870–12.354	0.079	5.919	0.871–40.232	0.069
High crowding group	3.619	0.990–13.227	0.052	9.482	1.526–58.901	0.016
CRP[a]	1.080	1.040–1.121	0.000	1.018	0.958–1.081	0.567
Serum creatinine	1.534	1.117–2.105	0.008	0.640	0.329–1.243	0.188
Albumin	0.067	0.028–0.157	0.000	0.128	0.034–0.485	0.002
Total cholesterol	0.989	0.977–1.000	0.052	1.013	0.999–1.027	0.072
PaO_2/FiO_2 ratio	0.989	0.984–0.994	0.000	0.994	0.988–1.000	0.055
PSI[b]	1.042	1.028–1.055	0.000	1.029	1.009–1.050	0.004
TFAD[c]	0.998	0.995–1.002	0.330	1.000	0.998–1.002	0.962

[a] CRP, C-Reactive Protein.
[b] PSI, Pneumonia Severity Index.
[c] TFAD, Time to First Antibiotic Dose.

Table 4 Associated factors with each time interval of TFAD.[a]

Variable	Unadjusted or	*p*-Value	Adjusted or	*p*-Value
TFAD < 8 h				
Low crowding group	Reference		Reference	
Intermediate crowding group	0.911	0.736	1.192	0.721
High crowding group	0.707	0.213	1.359	0.508
CRP[b]	0.904	0.000	0.892	0.001
Serum Creatinine	0.596	0.063	1.255	0.491
Albumin	2.237	0.001	0.728	0.455
Total cholesterol	1.006	0.047	0.991	0.110
PaO_2/FiO_2	1.003	0.007	1.003	0.056
PSI[c]	0.986	0.000	0.989	0.106
TFAD < 6 h				
Intermediate crowding group	1.064	0.811	1.181	0.697
High crowding group	0.892	0.655	1.469	0.335
CRP	0.921	0.000	0.918	0.001
Serum Creatinine	0.504	0.015	1.019	0.952
Albumin	1.896	0.002	0.689	0.315
Total cholesterol	1.007	0.009	0.995	0.340
PaO_2/FiO_2	1.003	0.007	1.002	0.118
PSI	0.987	0.000	0.989	0.055
TFAD < 4 h				
Intermediate crowding group	1.269	0.300	1.272	0.456
High crowding group	0.999	0.996	1.412	0.266
CRP	0.952	0.000	0.955	0.006
Serum Creatinine	0.681	0.026	0.906	0.647
Albumin	1.512	0.017	0.622	0.106
Total cholesterol	1.005	0.023	0.998	0.659
PaO_2/FiO_2	1.002	0.031	1.001	0.252
PSI	0.991	0.000	0.993	0.122
TFAD < 2 h				
Intermediate crowding group	1.200	0.490	1.295	0.414
High crowding group	1.239	0.406	1.612	0.124
CRP	0.970	0.008	0.993	0.629
Serum Creatinine	0.784	0.062	0.959	0.820
Albumin	1.880	0.001	1.311	0.354
Total cholesterol	1.005	0.077	0.998	0.549
PaO_2/FiO_2	1.004	0.000	1.004	0.004
PSI	0.989	0.000	0.998	0.597

Regression for each outcome (TFAD < 8 h, 6 h, 4 h, and 2 h) was controlled for crowding status, CRP, serum creatinine, albumin, total cholesterol, PaO_2/FiO_2, and PSI.

[a] TFAD, Time to First Antibiotic Dose.

[b] CRP, C-Reactive Protein.

[c] PSI, Pneumonia Severity Index.

knowledge, this is the first study to show the association between mortality in CAP patients and ED crowding.

In our study, we failed to show the association between a delay in first antibiotics delivery and ED crowding in contrast to the results of previous studies.[13–15] We believe that this result was derived from the quality improvement based on our previous study on implementing PSI in admission protocol, including the timely administration of antibiotics.[16]

Then, how does ED crowding influence mortality? This study was not designed to determine the exact mechanism responsible for this association, but some inferences can be made. First, ED crowding per se is a key mechanism responsible for higher mortality. In particular, since human and non-human resources are limited, ED crowding inevitably leads to a lower quality-of-care, which could cause significant morbidity and mortality,[11,12] especially in severe cases like CAP with higher PSI. Critical care constitutes a substantial proportion of ED care, and its proportion is growing. It is plausible that in the case of overcrowding in the ED, critical care in CAP patients, including the appropriate timing of fluid resuscitation, inotropics, or intubation might be delayed when clinical deterioration occurs in critically ill patients on boarding status or in waiting rooms even though antibiotics were delivered in a timely manner.[24–26] Medical errors and adverse events also might occur more frequently when the ED is crowding.[27–29]

Table 5 Cox proportional hazard regression analysis.

Variable	Harzard ratio	95% CI	*p*-Value
Low crowding group	Reference		
Intermediate crowding group	5.282	0.995—28.055	0.051
High crowding group	6.391	1.369—29.842	0.018
CRP[a]	1.003	0.956—1.053	0.891
Serum Creatinine	0.631	0.327—1.217	0.169
Albumin	0.230	0.079—0.668	0.007
Total cholesterol	1.006	0.997—1.016	0.180
PaO_2/FiO_2	0.994	0.989—1.000	0.062
PSI[b]	1.019	1.003—1.036	0.017
TFAD[c]	1.000	0.997—1.002	0.834

[a] CRP, C-Reactive Protein.
[b] PSI, Pneumonia Severity Index.
[c] TFAD, Time to First Antibiotic Dose.

Second, the crowding of the whole hospital could contribute to the increased mortality. The ED crowding could be caused partially by a shortage of inpatient beds in wards and intensive care units, and thus, high hospital occupancy might be one mechanism.

Another question is how the EDO in the early course of CAP affects long term mortality. There have been many studies, which emphasize the importance of early treatment of severe sepsis and its role in long term outcomes.[30,31] CAP is one of the most common severe sepsis, so same inference could be made in this context.

In the aggregate, there is some "dose—response" relation (or test for trend) in respect to the hazard ratio or adjusted odds ratio between tertile groups of overcrowding. However, in PSI IV group, it was not evident. One explanation is small sample size of non-survivors, and another intuitive explanation was that there would be some threshold of overcrowding, which mitigates the timely, proper and adequate management. Under that point, the impact of treatment could be same since human factor could buffer the mild shortage of human or non-human resources.

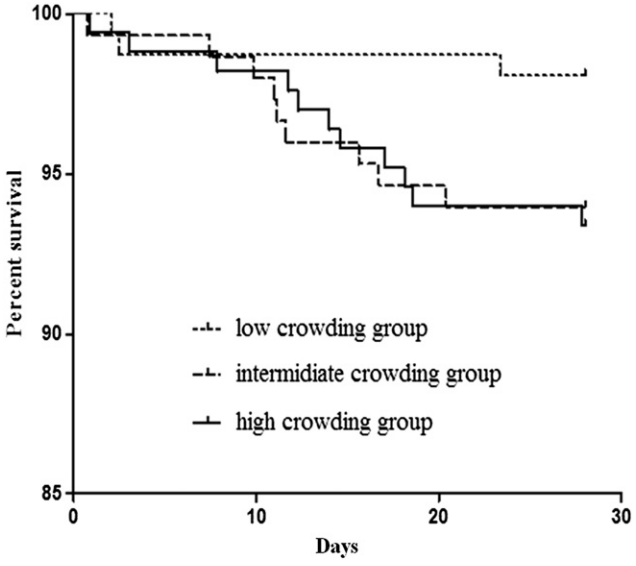

Figure 2 Survival proportions of each crowding group.

Limitations

The results of this study must be interpreted after considering some limitations. First, this result represents a single urban academic ED, and the results of this study cannot be generalized to other setting. Second, there were some missing mortality data even after making follow up phone call. The number, however, is small, so we do not think these missing data influenced our results significantly. Third, we did not collect data about the time to use of critical care such as the use of mechanical ventilator or about cases of medical error. Fourth, we did not design the study setting, which could show the dynamic stage of ED crowding. A recent study examined the ED crowding effect on waiting room, treatment and boarding times at 4 EDs.[32] In that study, crowding affected the waiting room time and the boarding time, but not the treatment time. Because effectiveness comparisons of ED interventions to decrease ED crowding for each ED crowding stage will be of interest to researchers, it is necessary to design studies to show the effect of ED crowding for each stages in the future. Lastly, with the small sample size, especially vis-à-vis primary outcome, it needs more larger study to confirm the association between the EDO and mortality in CAP patients.

Conclusion

CAP patients in the high crowding group showed significantly higher 28-day mortality rates than those in the low crowding group. However, there was no association between ED crowding and TFAD in this study.

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Conflict of interest

None declared.

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